Surgical management of mandibular condylar hyperplasia type 1

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This study compared outcomes of two surgical methods for patients diagnosed with active condylar hyperplasia type 1. Group 1 (n = 12) was treated with orthognathic surgery only, while group 2 (n = 42) was treated with high condylectomies, articular disc repositioning, and orthognathic surgery. There was no statistically significant difference between the two groups for maximum incisal opening, lateral excursions, and subjective jaw function before surgery. Group 2 showed more active presurgical mandibular growth ($P < 0.05$). At long-term follow up, no differences were found in lateral excursions and subjective jaw function. Group 2 showed a greater increase in maximum incisal opening ($P < 0.01$) and stability ($P < 0.05$) at long-term follow-up. All of the patients in group 1 grew back into skeletal and occlusal Class III relationships requiring secondary intervention, whereas all patients in group 2 remained stable in a Class I skeletal and occlusal relationship. Thus, patients with active condylar hyperplasia treated with high condylectomy, articular disc repositioning, and orthognathic surgery had stable, predictable outcomes compared with those treated with orthognathic surgery only. The high condylectomy effectively arrests disproportionate mandibular growth while maintaining normal jaw function.

Mandibular condylar hyperplasia (CH) is a pathological condition that causes overdevelopment of the condylar head and neck as well as the mandible, usually creating significant functional and aesthetic jaw and facial deformities (1–3). A number of different pathological entities can cause CH, with different effects on the dentofacial deformity. We have developed a simple classification to identify the various types of CH based on the frequency of occurrence, the types of jaw deformity created, and the surgical procedures necessary to get the best treatment outcomes.

CH type 1 is the most frequently occurring form and involves an accelerated growth rate of the "normal" growth mechanism of the mandibular condyle with relatively normal architecture of the condyle but elongation of the condylar head, neck, and mandibular body. This type, with a predominant horizontal growth vector, causes the mandible to grow forward of the maxilla, creating a Class III occlusal and skeletal relationship, although occasionally a vertical growth vector may occur (4). Type 1A is the bilateral form of CH with symmetric growth *(Figures 1a–c, 2a–c, 3a)* or asymmetric growth (one condyle growing faster than the opposite side). The less common unilateral form, type 1B, involves only one condyle, creating a progressively worsening facial asymmetry. CH type 1 causes mandibular prognathism (forward overdevelopment of the mandible). The onset of accelerated mandibular growth usually occurs during puberty, and the mandibular growth can continue into the mid 20s but is self-limiting. This mandibular overgrowth can cause major jaw and facial deformities.

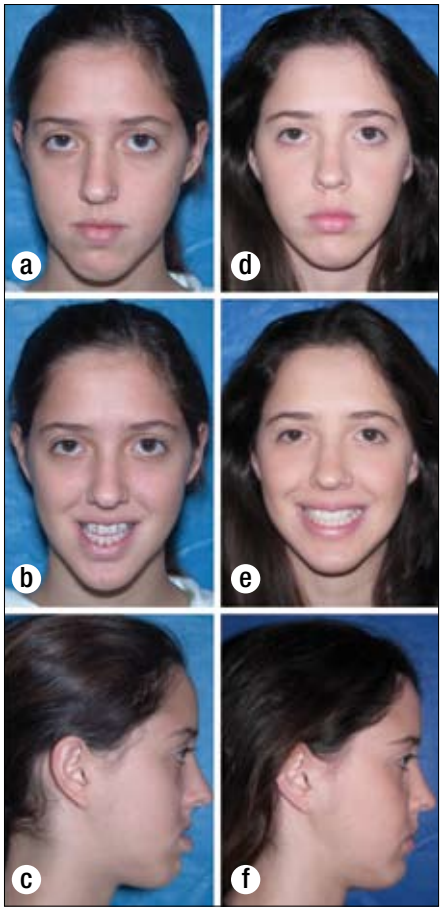

Figure 1. Case 1. **(a–c)** This 17-year-old female presented with symmetric mandibular prognathism due to active CH type 1. **(d–f)** The patient is seen 24 months after surgery, demonstrating good facial balance.

CH type 2 occurs unilaterally and involves enlargement of the condylar head; usually the condylar neck increases in thickness and the vertical height of the mandibular ramus and body in-

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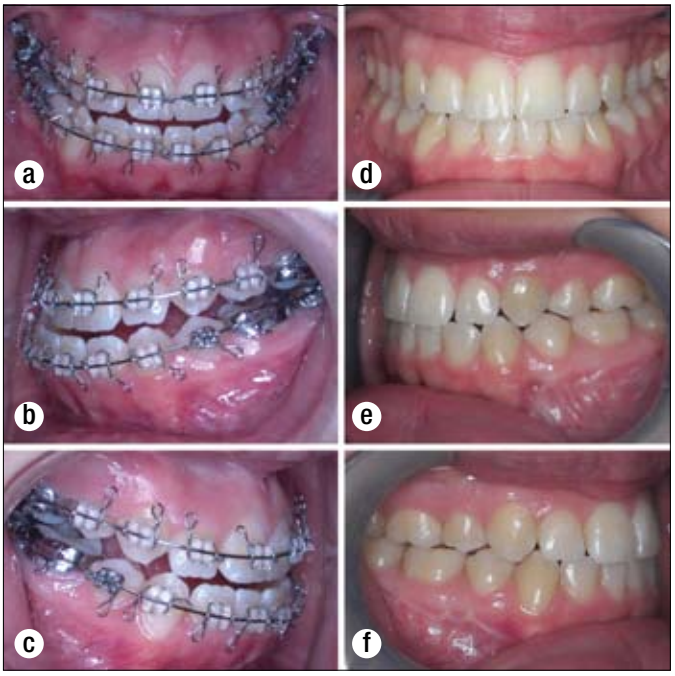

Figure 2. Case 1. **(a–c)** The patient demonstrates a Class III occlusion before surgery. **(d–f)** At 24-month follow-up, the patient shows a very stable Class I occlusion.

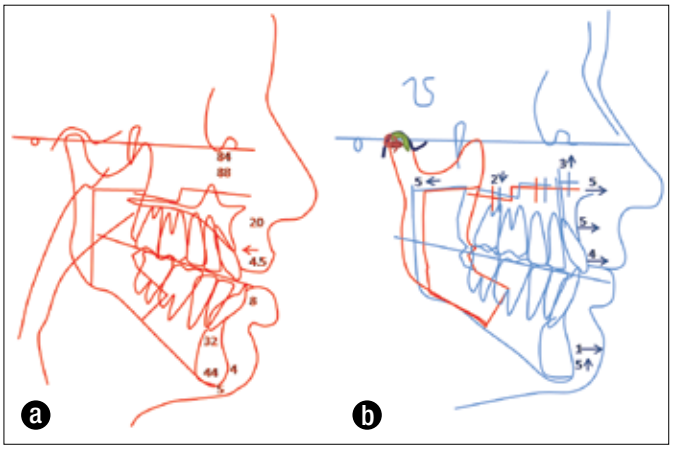

Figure 3. Case 1. **(a)** The presurgical lateral cephalometric tracing demonstrates a skeletal and occlusal Class III relationship. **(b)** The surgical prediction tracing illustrates the procedures performed, including bilateral high condylectomies, repositioning of the articular discs, bilateral mandibular ramus osteotomies to set the mandible posteriorly into a proper relationship, and maxillary advancement.

creases on the ipsilateral side, often accompanied by a compensatory downward growth of the ipsilateral maxilla (4). CH type 2 can occur at any age and is not self-limiting. CH type 2 can be caused by an osteochondroma, osteoma, or other rare forms of condylar enlargement (i.e., benign or malignant tumors of the mandibular condyle, hemifacial hypertrophy, etc.).

CH type 1 occurs much more frequently than clinicians realize and is the most common form of CH. Failure to recognize this pathological entity can result in unfavorable functional and aesthetic treatment results following orthodontics and orthognathic surgery if the CH factors are ignored. However, this condition can be surgically treated effectively with highly predictable outcomes (4).

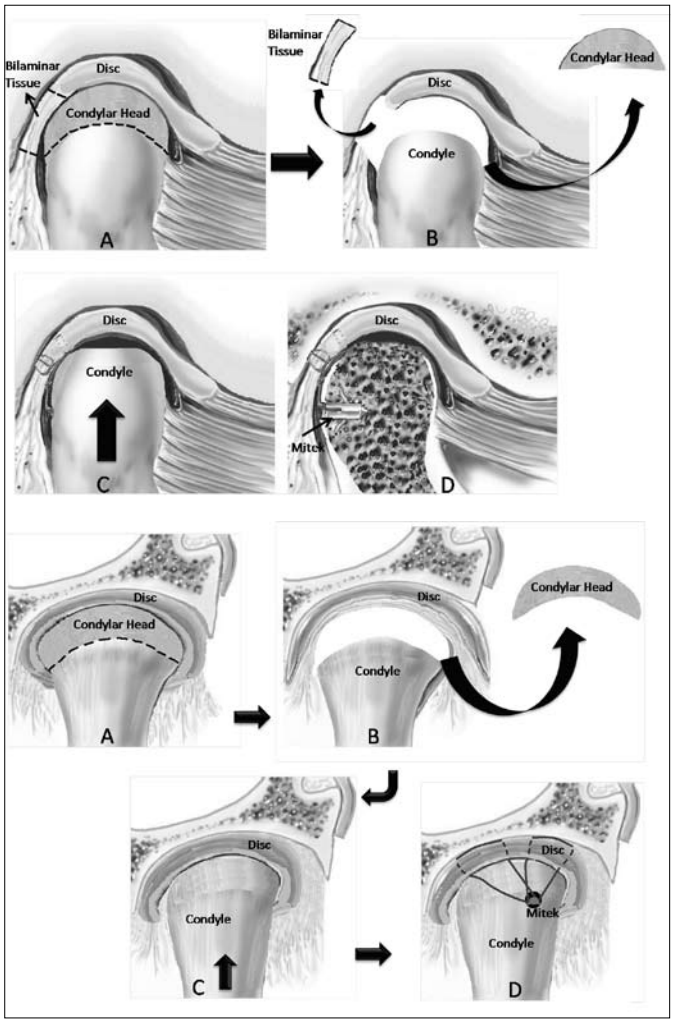

Figure 4. High condylectomy and disc repositioning surgery, with both lateral (top) and posterior (bottom) views. **(a, b)** The high condylectomy involves removal of the top 3 to 5 mm of the condylar head including the lateral and medial poles. **(c, d)** The TMJ articular disc has been repositioned over the condylar stump and secured in position using a Mitek mini anchor. Cortical bone will reform over the head of the condyle.

Although we have previously demonstrated that mandibular setback surgery for correction of mandibular prognathism in nongrowing patients without active CH is a very stable procedure (5), numerous studies have reported relapse for mandibular setback ranging from 20% to 91% of the amount of posterior surgical movement (6–14). It is likely that this high percentage of relapse is in part due to unrecognized and untreated active CH type 1. Understanding the etiology of CH, nature of the deformity, clinical presentation, options available for treatment, and timing of treatment is required for achieving optimal treatment outcomes.

In 1979, Wolford (15, 16) developed a technique to predictably eliminate mandibular growth in CH type 1 by performing high condylectomies along with simultaneous orthognathic surgery to correct the associated jaw deformity. The high condylectomy arrests the excessive and disproportionate growth of the mandible by surgically removing one of the important mandibular growth sites and the site responsible for the CH type 1 pathological growth process (4) *(Figure 4)*.

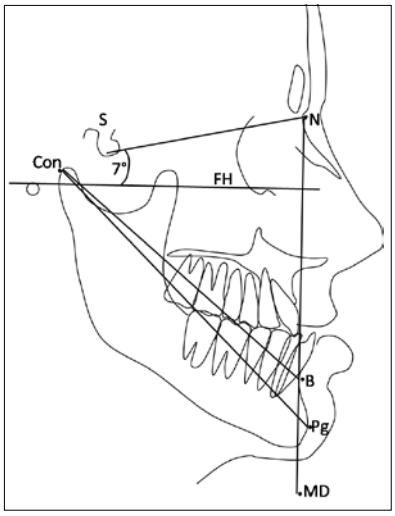

Figure 5. Cephalometric tracings identifying important landmarks used in this study: S, sella, midpoint of sella turcica; N, nasion, nasofrontal suture; FH, Frankfort horizontal plane, plane constructed at 7° to the sella nasion line through the infraorbital rim; Con, condylion, the most posterosuperior point on the posterior condylar head; B, point B, the most posterior point on the anterior mandible between the alveolar bone and chin; Pg, pogonion, most anterior point on the chin; MD, mandibular depth, the angle formed by the FH and nasion–point B line.

Ninety-eight percent of facial growth is completed by age 15 in females and by age 17 or 18 in males. During the pubertal growth years, the mandible grows and lengthens from condylion to point B *(Figure 5)* at a yearly growth rate of 1.6 mm for females and 2.2 mm for males (17). Growth at a significantly accelerated rate or for a prolonged postpubertal time interval usually indicates active CH (4).

In a previous study (4), we evaluated 37 patients with documented active CH type 1 divided into two groups based on treatment. Group 1 patients (n = 12) were treated with orthognathic surgery only, and group 2 patients (n = 25) were treated with high condylectomy, articular disc repositioning, and orthognathic surgery. Group 1 patients were older than group 2 patients (17.5 years vs 16.7 years), but group 2 had a greater excessive mandibular growth rate (4.5 mm) during the year prior to surgery than group 1 (2.4 mm). After surgery, with an average of >5 years of follow-up, group 1 had additional mandibular growth of 3.5 mm compared with 0.8 mm in group 2, a statistically significant difference. Jaw function (MIO and excursive movements) were essentially equal after surgery in both groups. After surgery, all patients in group 1 grew into Class III skeletal and occlusal relationships, requiring additional surgical intervention for correction. All patients in group 2 maintained a stable Class I skeletal and occlusal relationship (4).

In this article, treatment outcomes and long-term follow-up stability from these two surgical methods are compared in a larger patient population with active CH type 1.

PATIENTS AND METHODS

The treatment records of patients diagnosed with active CH, treated by the senior author (Wolford) prior to 2000, were retrospectively analyzed. There were five criteria for inclusion in the study: 1) confirmed active CH type 1 based on serial clinical and radiographic evaluations (serial lateral cephalograms and lateral temporomandibular joint [TMJ] cephalometric tomograms); 2) bilateral or unilateral high condylectomy to remove the active growth center and articular disc repositioning for group 2; 3) orthognathic surgery to correct the associated dentofacial deformity; 4) at least 2 years of postsurgical follow-up; and 5) adequate clinical and radiographic records for analysis.

Fifty-four patients (36 females, 18 males) met the inclusion criteria. Group 1 patients (n = 12) were treated with orthognathic surgery only, while group 2 patients (n = 42) had high condylectomy procedures, articular disc repositioning, and orthognathic surgery.

All patients underwent standardized clinical and radiographic examination at the following intervals: initial consultation (T1), immediate presurgery (T2), immediate postsurgery (T3), and longest follow-up (LFU) (T4). All clinical examinations were performed by a single clinician. Objective evaluation of TMJ function, maximum incisal opening (MIO), and lateral excursions were recorded on all patients. For group 2 patients, subjective evaluations were performed using numerical analog scales to assess TMJ pain, jaw function, and diet.

Lateral cephalograms at T1, T2, T3, and T4 were traced and superimposed to calculate presurgical growth change (T2 – T1), surgical change (T3 – T2), and long-term stability (T4 – T3). The lateral cephalometric radiographs were assessed on all patients for 1) mandibular depth (Frankfort horizontal plane – nasion–point B line); 2) condylion-pogonion length; and 3) condylion–point B length *(Figure 5)*. A calibration error test was performed for

Table 1. Characteristics of patients treated for mandibular condylar hyperplasia type 1*

Group	N	Sex	Age (years) (range)	Affected side	Follow-up (range)	
					Presurgical (months)	Postsurgical (years)
1	12	8 F, 4 M	17.5 (13 to 25)	12 bilateral	12.5 (5 to 43)	5.6 (2 to 11.2)
2	42	28 F, 14 M	16.6 (13 to 24)	24 bilateral 18 unilateral	12.1 (4 to 45)	5.1 (2 to 16.9)

*Group 1 was treated with orthognathic surgery only; group 2 was treated with high condylectomies, articular disc repositioning, and orthognathic surgery.

Table 2. Objective evaluation of patients treated for mandibular condylar hyperplasia type 1*

Group	Time period	Maximum incisal opening (mm) (range)	Lateral excursions (mm) (range)
1 (n = 12)	Presurgical (T2)	46.8 (26 to 53)	7.0 (5 to 9)
	Longest follow-up (T4)	46.7 (43 to 50)	7.5 (6 to 10)
2 (n = 42)	Presurgical (T2)	40.2 (26 to 49)	7.7 (3 to 10)
	Longest follow-up (T4)	49.5† (31 to 62)	7.8 (4 to 12)

*Group 1 was treated with orthognathic surgery only; group 2 was treated with high condylectomies, articular disc repositioning, and orthognathic surgery.

†Statistically significant at $P < 0.01$ level.

each parameter in 10 different cephalograms. The calibration showed a high correlation ($r > 0.96$) for both intra- and interoperator examiners, with a standard error of <0.45 for each parameter. Student's t test was utilized to detect differences between groups and between time intervals, at a significance level of $P \leq 0.05$.

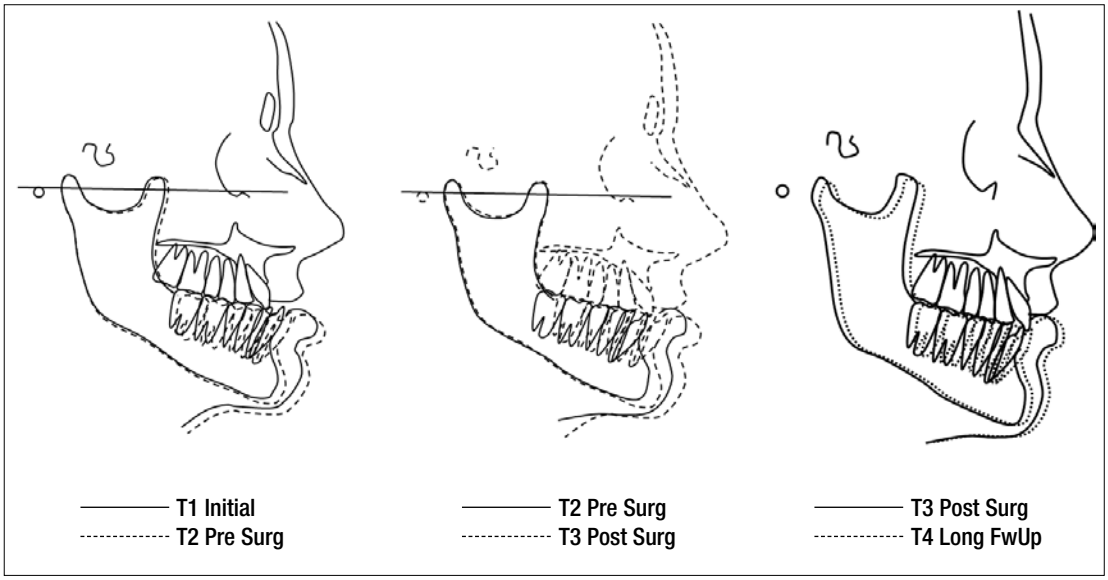

Figure 6. Presurgical, surgical, and postsurgical changes in groups 1 and 2. Presurgically (T2 – T1), both groups showed a similar growth pattern, with group 2 being greater. Both groups received a similar treatment and achieved equal results immediately after surgery (T3 – T2). At follow-up (T4 – T3), group 1 showed a statistically significantly higher postsurgical growth rate than group 2. T1 indicates initial cephalogram; T2, immediate presurgical cephalogram; T3, immediate postsurgical cephalogram; T4, longest follow-up cephalogram.

RESULTS

Patient characteristics are shown in *Table 1.* Because the statistical analysis showed no significant sex differences or differences between unilateral and bilateral patients, the samples were pooled for subsequent analyses.

Objective clinical data

MIO and LE values before surgery (T2) and at LFU (T4) are shown in *Table 2.* Group 1 showed no significant change in MIO ($P > 0.05$), while group 2 showed a statistically significant increase ($P < 0.01$). There was no significant difference between the two groups when comparing lateral excursions before surgery and at LFU.

Subjective clinical data

The form that gathers subjective clinical data is used only for patients that have TMJ surgery; it is not used for orthognathic surgery cases without TMJ symptoms or known TMJ pathology. None of the patients in group 1 had TMJ dysfunction or reported pain before surgery or at LFU, so subjective data were available only for group 2 patients. As shown in *Table 3,* TMJ pain was not a common symptom before surgery, and no statistically significant difference was found when comparing presurgical and LFU values. The average jaw function score improved slightly, from 3.4 to 2.1, but the difference was not statistically significant. No significant dietary restrictions were reported by any patient before surgery or at LFU.

Cephalometric data

All patients in group 1 grew back into skeletal and occlusal Class III relationships and required secondary surgical intervention to correct the resultant deformity. Only one patient in group 2 required secondary surgery, and that involved repeat maxillary surgery for

Table 3. Subjective evaluation of group 2 patients treated for mandibular condylar hyperplasia type 1 with high condylectomies, articular disc repositioning, and orthognathic surgery

	Score (range)		
Time period	TMJ pain	Jaw function	Diet
Presurgical (T2)	0.6 (0 to 2)	3.4 (0 to 5)	0.6 (0 to 1)
Longest follow-up (T4)	0.2 (0 to 2)	2.1 (0 to 5)	0.4 (0 to 1)

*All evaluations used a scale of 0 to 10. For temporomandibular joint (TMJ) pain, 0 = no pain, 10 = worst pain; for jaw function, 0 = normal function, 10 = no function; for diet, 0 = no restriction, 10 = liquids only.

Table 4. Cephalometric evaluation of patients treated for mandibular condylar hyperplasia type 1*

		Result (range)		
Group	Difference	Mandibular depth (degrees)	Co-Pg (mm)	Co-B (mm)
1 (n = 12)	T2 – T1	1.35 (–1 to 5)	2.4 (1 to 5)	2.3 (1 to 5)
	T3 – T2	–4.1 (–1 to –7)	–4.7 (–1 to –9)	–4.8 (–3 to –12)
	T4 – T3	2.8† (0 to 5)	3.5‡ (1 to 11)	3.6‡ (2 to 7)
2 (n = 42)	T2 – T1	2.1 (1 to 5)	4.6† (1 to 14)	3.7† (1 to 11)
	T3 – T2	–4.8 (–1 to –7)	–5.4 (–1 to –8)	–5.5 (–1 to –10)
	T4 – T3	0.6 (0 to 2)	0.6 (0 to 3)	0.3 (0 to 2)

*Group 1 was treated with orthognathic surgery only; group 2 was treated with high condylectomies, articular disc repositioning, and orthognathic surgery.

†Statistically significant at $P < 0.05$ level, comparing group 1 with group 2.

‡Statistically significant at $P < 0.01$ level, comparing group 1 with group 2.

T1 indicates initial consultation; T2, immediate presurgery; T3, immediate postsurgery; T4, longest follow-up; Co-Pg, condylion-pogonion length; Co-B, condylion–point B. Mandibular depth is the Frankfort horizontal plane – nasion–point B line.

correction of postsurgical transverse maxillary relapse; the mandible, however, was stable at LFU.

For mandibular depth, there was no significant difference between the two groups when comparing growth before surgery and the surgical change. However, there was a significant difference ($P < 0.02$) between the two groups in the amount of postsurgical change (T4 – T3), where some change may be related to occlusal splint removal at 4 to 6 weeks postsurgery with slight forward and upward rotation of the mandible and settling of the occlusion *(Figure 6, Table 4).* Condylion-pogonion length and condylion–point B presurgical measurements showed a statistically significant difference ($P < 0.05$) between the two groups, with group 2 presenting greater active growth. A nonstatistically significant difference was seen when comparing the surgical changes.

DISCUSSION

CH type 1 results in a horizontal overdevelopment of the mandible and is usually termed symmetrical or deviated prognathism, laterognathia, or mandibular hyperplasia. However, the basic cause of many mandibular prognathic cases is excessive mandibular growth originating in the mandibular condyles. CH type 1 occurred twice as often in females than in males in our study and occurred more often bilaterally rather than unilaterally. Patients usually demonstrate a Class I or mild to moderate Class III skeletal and occlusal relationship prior to onset of CH at puberty and develop into a Class III or worse Class III relationship, respectively, as the accelerated growth progresses *(Figures 1a–c, 2a–c, 8a–c, 9a–c).* CH type 1 rarely occurs in skeletal and occlusal Class II patients. Common clinical and radiographic characteristics are listed in *Table 5.*

The differential diagnosis for CH type 1 includes 1) maxillary hypoplasia; 2) mandibular prognathism without CH (patients start out as skeletal Class III in early childhood and maintain harmonious growth between maxilla and mandible, with cessation of growth at the normal ages); 3) dislocation of condyles anterior to the articular eminence; 4) dental interferences or habitual posturing causing anterior positioning of the mandible; 5) acromegaly; 6) macroglossia; 7) congenital or acquired facial asymmetry unrelated to the TMJ; and 8) other TMJ pathology such as osteochondroma, osteoma, or contralateral condylar resorption.

It is important to identify the type of pathology and growth pattern in mandibular CH and to determine if growth is in the active or inactive state.

Growth patterns

The specific growth pattern of the condyle(s), in terms of magnitude, rate, and direction, can influence the timing of surgery and the type of corrective surgical procedures required. CH type 1A with bilateral involvement but an asymmetric differential condylar growth rate or CH type 1B (unilateral) will develop mandibular transverse asymmetry toward the slower growing or nongrowing side. CH type 1 usually presents with a horizontal growth vector. A normal condyle is approximately 15 to 20 mm in mediolateral dimension and 8 to 10 mm wide anteroposteriorly (33). Although the condyle usually retains a relatively normal architecture, an increased length of the condylar head, neck, and mandibular body is commonly seen (2, 3, 19–21).

Table 5. Common clinical and radiographic characteristics observed in bilateral, symmetrically growing condylar hyperplasia type 1 patients

1. Increased length of the condylar head and neck, without a significant volumetric increase in the size of the condylar head
2. Mandibular growth occurring at an accelerated rate
3. Mandible continuing to grow beyond the normal growth years
4. Worsening Class III skeletal and occlusal relationship
5. Worsening aesthetics
6. Obtuse gonial angles
7. Decreased angulation of lower incisors and increased angulation of upper incisors (dental compensations)
8. Decreased vertical height of the posterior mandibular body
9. High mandibular plane angle
10. Narrow anteroposterior and mediolateral dimensions of the rami and symphysis in more severe cases

Additional characteristics in asymmetric cases:

1. TMJ articular disc displacement and arthritis on the contralateral side as a result of increased loading of that joint caused by the condylar hyperplasia on the opposite side
2. Worsening facial and occlusal asymmetry, with the mandible progressively shifting toward the contralateral side
3. Unilateral posterior cross-bite on the contralateral side
4. Transverse bowing of the mandibular body on the ipsilateral side
5. Transverse flattening of the mandibular body on the contralateral side

CH type 1 is often undiagnosed because of the general lack of understanding of clinicians that this aberrant prolonged condylar growth pattern can create mandibular prognathism. It is often perceived that mandibular prognathism is associated with growth disturbances in the mandibular body. Although the mandibular body and alveolar bone are affected, the primary stimulus creating the deformity is the result of the accelerated and prolonged "normal" growth mechanism of the mandibular condyle(s). The high condylectomy stops forward growth of the mandible, with only normal appositional growth remaining at pogonion and vertical alveolar growth if the surgery is performed before normal facial growth is completed (4, 15).

Determining active growth

Active CH type 1 growth can usually be determined by worsening functional and aesthetic changes with *serial assessments* (preferably at 6- to 12-month intervals) consisting of 1) clinical evaluation; 2) dental analysis with orthodontically trimmed models or articulator-mounted models in centric relation; and 3) radiographic evaluation by superimposition including lateral cephalometric radiographs, frontal cephalometric radiographs (particularly helpful in unilateral CH cases), and

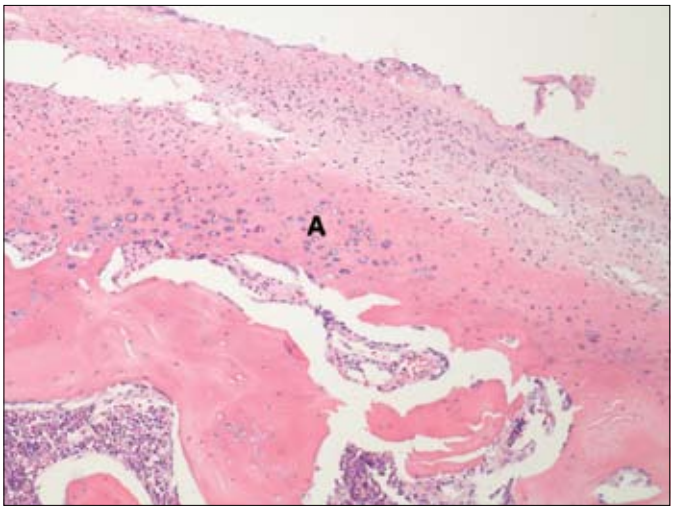

Figure 7. The condylar histology of CH type 1 appears similar to normal growing condyles. There can be prominences of cartilage-producing cells (A) at the lower border of the proliferative layer. Hematoxylin and eosin stain, ×100.

lateral cephalometric tomograms that include the TMJ, mandibular ramus, and body.

Bone scanning with technetium-99m pyrophosphate or technetium-99m methylene diphosphonate has been advocated to detect active growth in the condyle (22–26). This may be most effective in unilateral cases, especially if applied after the normal growing years when condylar growth should have ceased. However, the senior author has not found bone scans diagnostic in most cases and therefore does not recommend the use of bone scans for CH type 1 patients. Normal condyles have increased uptake with bone scanning, and the rate of accelerated growth may not be detectable. Furthermore, in asymmetric CH types 1A and 1B, the contralateral TMJ may present with arthritic condylar changes, a displaced articular disc, and associated inflammation that may also present with increased isotope uptake, rendering the bone scan inconclusive. In our experience, we have found bone scans to be inconclusive in younger patients, in patients with slow-growing CH, and in patients with coexisting disc displacement on the contralateral side. Hand-wrist films are of no value in CH since the mandible can continue to grow well beyond the normal growth years.

Etiology and histology

The identification of sex hormone receptors in and around the TMJ and the pubertal onset of CH type 1 strongly suggest a hormonal influence in the etiology. Trauma (27–31), infection (1, 27, 32, 33), heredity (3, 34–38), intrauterine factors (28, 39, 40), and hypervascularity (38, 41) have also been implicated as causative factors for CH type 1 and type 2. Approximately one third of bilateral CH type 1 cases have a familial history (34).

Histological observations of CH type 1 condyles may appear very similar to normal bony architecture. In other cases, the proliferative layer may demonstrate greater thickness in some areas and less in others, and cartilage-producing cells may be prominent at the lower border *(Figure 7).* In some regions, the cartilage may be thickened, actively generating, and replaced by new bone. The activity of the proliferative layer may regulate the rate at which the condyle and condylar neck (which is formed from the condyle by remodeling) will grow. In normal condyles, the formation of cartilage from the proliferative layer and the replacement of cartilage by bone cease by approximately 20 years of age. The marrow cavity is entirely occluded from the remaining cartilage by the closure of the bone plate. The inability of this plate to close in the presence of an active proliferative cartilage layer may be a major etiological factor in CH type 1 and may correlate to our observation that cessation of growth related to CH type 1 may not occur until the mid 20s (4). Conditions that initiate excessive accelerated unilateral mandibular growth after the age of 20 are most often related to an osteochondroma, osteoma (CH type 2), or other rare types of proliferative condylar pathology.

Treatment options

Cases with arrested CH type 1 (in which the abnormal condylar growth has stopped and is now stable) can usually be treated with routine orthodontics and orthognathic surgery. The basic orthodontic goals for CH type 1 are to align and level the teeth over the basal bone in each jaw independently and remove dental compensations, regardless of the magnitude of skeletal and dental malalignment. However, active CH type 1 cannot be controlled with orthodontics or orthopedic mechanics. There are three surgical treatment options for active CH type 1 patients. Based on our extensive experience and supported by the results of this study, our treatment choice is *option #1.*

Treatment option #1. Surgically eliminate further mandibular CH growth with a high condylectomy (removing 3 to 5 mm of the superior aspect of the condylar head including the medial and lateral poles) *(Figures 3b, 4, 10b)* and simultaneous orthognathic surgery (4); this was the treatment used for all patients in group 2. The TMJ articular disc is repositioned and stabilized to cover the articulating surface of the "new" condyle. Cortical bone reforms over the top of the condyle. We prefer to use the Mitek mini anchor (Mitek Inc., Norwood, MA) with artificial ligaments to stabilize the articular disc to the condyle (42–44). The high condylectomy and disc repositioning procedure can be combined with one-stage simultaneous orthognathic surgery to correct the jaw deformity that will include mandibular ramus osteotomies and, when indicated, maxillary osteotomies *(Figures 3b, 10b)* (4, 45, 46). In CH type 1B cases, the contralateral TMJ may demonstrate a displaced articular disc and arthritis secondary to the physiological overload of the joint created by the ipsilateral CH. In these cases, the contralateral disc must be repositioned and stabilized with a Mitek anchor for joint stability at the same operation. Following this protocol, highly predictable outcomes can be achieved *(Figures 1d–f, 2d–f, 8d–f, 9d–f).* Less experienced surgeons can perform this procedure in two surgical stages: the high condylectomy and disc repositioning can be performed as stage 1, followed by orthognathic surgery at a second stage.

Treatment option #2. Defer corrective surgery until growth is complete, which often means delaying surgery until the mid 20s. Consequently, the patient may suffer from functional problems (mastication, speech), worsening facial disfigurement, pain, and psychosocial stigmas associated with a severe facial

deformity (3, 13). Additionally, the magnitude of the deformity, if allowed to fully manifest by this delay in treatment, may preclude an optimal result later. CH type 1 growth may result in severe deformation of the mandible as well as compensatory changes in the maxilla, dentoalveolus structures, and associated soft tissue structures, significantly compromising the subsequent surgical treatment outcome.

Treatment option #3. Perform only orthognathic surgery during active CH type 1 growth, with consideration for overcorrection of the mandible. The accelerated mandibular condylar growth will continue after surgery, and repeat surgery will be needed if the estimated overcorrection is greater or less than necessary. Early intervention may benefit the patient, relative to function, aesthetics, and psychosocial concerns. With this option, surgery is best performed after the majority of maxillary growth is complete (15 years in females and 17 to 18 years in males) to help facilitate the estimation of overcorrection necessary. In our study, group 1 patients were treated with only orthognathic surgery and were placed in the best occlusion fit at the time of surgery. All the patients in group 1 grew into a Class III occlusal and skeletal relationship and required additional surgical intervention.

When orthognathic and TMJ surgeries are performed at one operation, we recommend using the sagittal split ramus osteotomy for the mandible since it provides positional control of the ramus and condyle, maintains maximal soft tissue attachment and vascularity to the proximal segment, and allows easy application of rigid fixation. The high condylectomy procedure will alter the position of the mandible to the maxilla since vertical height of the condyle is removed. Therefore, following the TMJ surgery, we perform the mandibular ramus sagittal split osteotomies with rigid fixation to place the mandible into its predetermined final position relative to the unoperated maxilla.

Figure 8. Case 2. **(a–c)** This 21-year-old woman presented with symmetric mandibular prognathism due to active CH type 1. **(d–f)** The patient 52 months after surgery, demonstrating good facial balance and stability.

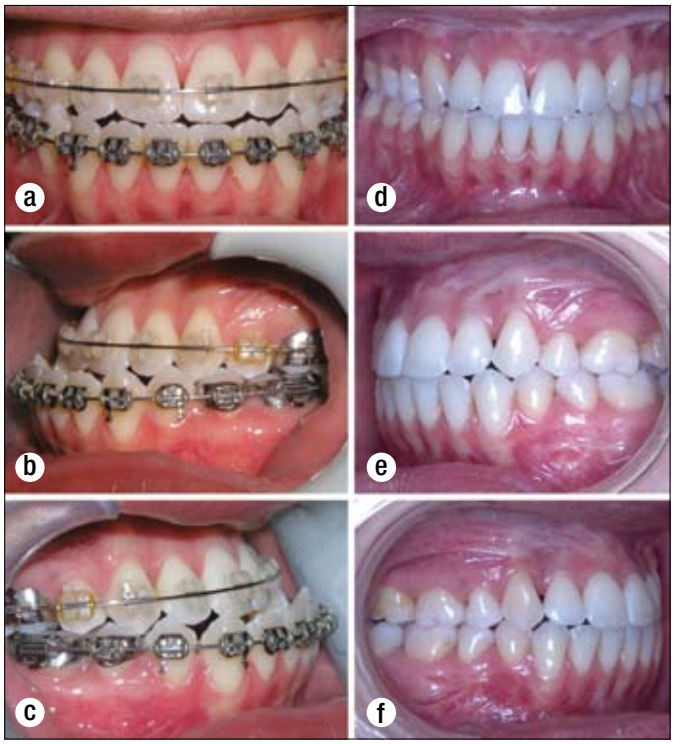

Figure 9. Case 2. **(a–c)** The patient demonstrates a Class III occlusion before surgery. **(d–f)** At 52-month follow-up, the patient shows a very stable Class I occlusion.

Thus, it doesn't matter how much the mandible is displaced with the high condylectomy procedure; the mandible is still placed in the same final position. Then the maxillary osteotomies with application of rigid fixation are completed *(Figures 3b, 10b)* as well as any other additional procedures (i.e., turbinectomies, nasoseptoplasty, genioplasty, rhinoplasty, etc.) (4, 45, 46). After surgery, intermaxillary fixation is not used, but light elastics are applied (usually in a slight Class III vector initially) to control the occlusion, minimize edema in the joint(s), and provide support to the muscles of mastication. The elastics may be used for 1 to 2 weeks or longer as required for occlusal control and to initiate postsurgical orthodontic finishing mechanics (47). Other mandibular ramus osteotomy techniques such as the inverted L or vertical ramus osteotomies require increased stripping of periosteum and musculature, with greater risk for vascular compromise of the proximal segment, besides causing difficulties with positional control of the condyle and the necessity for intermaxillary fixation, as well as stability problems if maxillary osteotomies are required.

Surgical correction of bilateral CH type 1 (symmetric or asymmetric) with simultaneous orthognathic surgery can predictably be performed from the age of 14 years in females and 16 years in males. The vector of facial growth will change to a downward and backward direction because the anteroposterior mandibular growth is arrested, but the maxillary vertical alveolar growth will continue until maturation. In unilateral cases, we recommend delaying surgery until the age of 15 years for females and 17 to 18 years for males, since most of the normal facial growth is complete by that time. A unilateral high condylectomy will arrest growth on the operated side, but normal

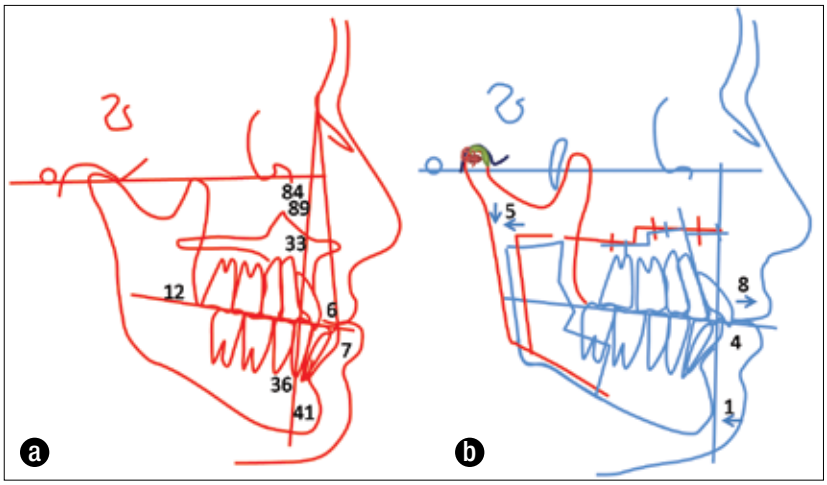

Figure 10. Case 2. **(a)** The presurgical lateral cephalometric tracing demonstrates a skeletal and occlusal Class III relationship of the ramus. **(b)** The surgical prediction tracing illustrates the surgical procedures performed, including bilateral high condylectomies, repositioning of the articular discs, bilateral mandibular ramus osteotomies to set the mandible posteriorly into a proper relationship, and maxillary advancement.

growth can continue on the contralateral side and could cause development of facial and occlusal asymmetry after surgery if the surgery is performed while normal growth is still occurring.

CONCLUSIONS

The results from our study demonstrate that the high condylectomy (with removal of the top 3 to 5 mm of the condylar head) in the treatment of active CH type 1 patients arrests the abnormal growth and provides highly predictable long-term outcomes. All the group 1 patients (n = 12) with active CH treated with orthognathic surgery only grew into Class III occlusal and skeletal relationships and required additional surgical intervention to correct the resultant malocclusion. On the other hand, the patients in group 2 (n = 42) remained stable in a Class I skeletal and occlusal relationship, with the exception of one case that involved postsurgical maxillary transverse relapse and the development of bilateral posterior cross-bites, requiring a secondary maxillary surgical intervention, but the mandible remained stable. None of the patients in group 2 demonstrated any significant postsurgical mandibular growth, except for expected appositional growth at pogonion. Long-term follow-up revealed no undesirable changes in the subjective and objective jaw function, with maintenance of MIO and lateral excursion values. No patient reported any significant TMJ pain or dietary restrictions at LFU.

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